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STUDIES WITH
STRAIN GAUGE
PLETHYSMOGRAPHY

by

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Illustration Department
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The thesis is based on the following papers:

- I. Englund, N., Hallböök, T. & Ling, L.
The validity of strain gauge plethysmography.
Scand. J. Clin. Lab. Invest 29 (2) 155-158 1972
- II. Dahn, I. & Ling L.
Venous resistance in human calf during external pressure
and/or venous stasis.
Acc. for publication Wasa nr 3 1973
- III. Dahn, I. & Ling L.
Venous resistance during external pressure and/or venous
stasis during reactive hyperaemia in human calf.
Acc. for publication Scand. J. Clin. Lab. Invest. 1973
- IV. Ling, L.
The effect of external pressure and elevation on venous
function in normal and thrombotic leg.
Angiology 23 453-463 1972
- V. Hallböök, T. & Ling L.
Resting blood flow at deep venous thrombosis of the leg.
Acta Chir. Scand. 138 581-584 1972

In the text, these papers are referred to by their Roman numerals.

INTRODUCTION

Towards the end of the 1950s, peripheral vascular surgery began at the surgical department in Lund. The angiographic examination was of great value, but needed to be completed with blood flow recording to ensure an adequate management of the patients with vascular disorders. At the clinical department of physiology in Lund, Dahn developed a water plethysmograph for clinical use. The plethysmograph was large and cumbersome. By making use of the strain gauge plethysmograph (Whitney 1953), Hallböök (1970) was able to make blood flow recordings even outside the laboratory. Some theoretical objections, however, have been raised against the strain gauge plethysmograph (Ardill et al. 1968), which is the subject in the first part of the thesis.

The second part deals with the relation between blood pressure and blood flow. During the mid-1960s, great interest was focused on the special blood flow situation of the gangrenous extremity by members of the department of surgery and clinical physiology in Lund and of the department of clinical physiology at Bispebjerg, Copenhagen. The effect of low position and the value of treatment with hypertensive drugs were noted (Dahn et al. 1967). The hypothesis of critical closing pressure (Burton 1951) was discussed with results from experiments with external pressure and venous stasis (Dahn et al. 1967).

In the thesis, these problems are again discussed with special regard to the resistance to the blood flow in the veins.

During plethysmographic investigations, Dahn (1967) noticed that patients with deep venous thrombosis differed from the other patients. These differences could be used for diagnosing deep venous thrombosis (Dahn & Eiriksson 1968; Hallböök & Göthlin 1971; Hallböök & Ling 1973). The special blood flow situation of the deep venous thrombosis is discussed in the third and last part of the thesis.

THE VALIDITY OF STRAIN GAUGE PLETHYSMOGRAPHY

The strain gauge plethysmograph (Whitney 1953) is simple and easy to handle, as it consists only of a mercury-filled latex gauge with a diameter of a few millimeters. The method has been further simplified by electrical calibration (Brakké & Vendrik 1966; Hallböök *et al.* 1970). Theoretical objections, however, have been raised against it (Ardill *et al.* 1968). It does not record the direct increase in the volume, but records the increase in the circumference of the segment, which is then converted to increase in volume.

A unique possibility to test the validity of the strain gauge plethysmograph was found during regional perfusion of the human leg. It was possible to vary the inflow in the limb and at the same time to record the blood flow in the calf with the instrument.

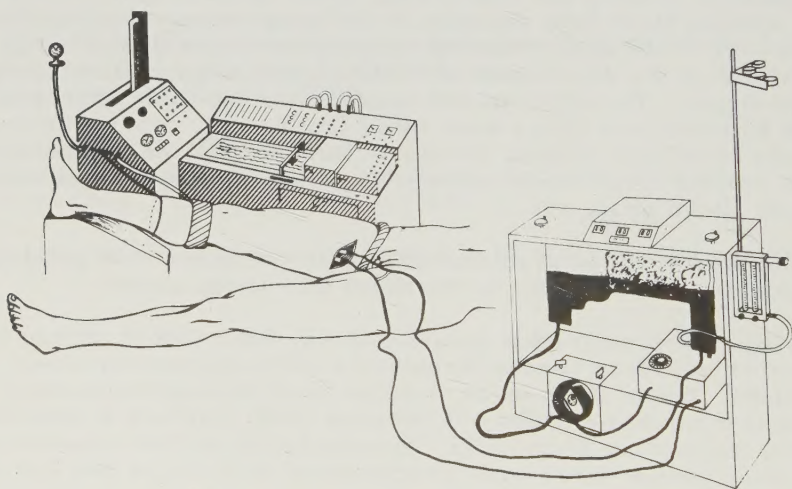


Fig. 1: Silver cannulas were inserted into the femoral artery and vein just below the inguinal ligament. The venous blood was drained to the bubble oxygenator of the extracorporeal circuit. The blood was then pumped over the heat exchanger to the femoral artery (Englund *et al.* 1971).

The temperature of the perfusate was 42°C (Krements & Ryan 1972).

The strain gauge plethysmograph was placed 15 cm below the knee joint. The collection cuff was placed below the knee, because the area above the knee was sterile washed.

The inflow was varied by changing the speed of the pump. At every change of the inflow, the flow in the calf was recorded with the strain gauge plethysmograph.

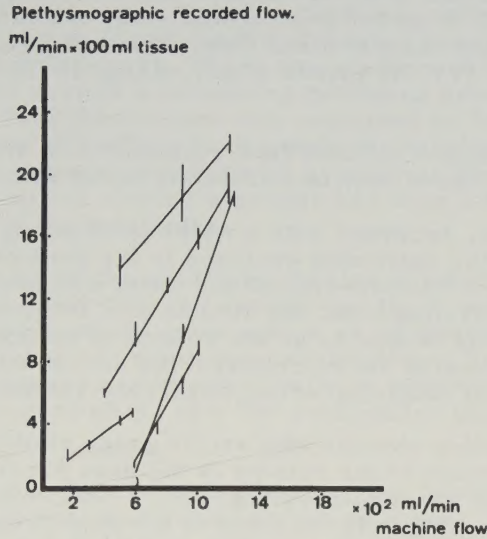


Fig. 2: The relation between machine flow and peripheral plethysmographic recorded flow. On the abscissa is the machine flow (ml/min); on the ordinate, the plethysmographic recorded flow (ml/min $\times 100$ ml tissue) plotted in mean values and S.D.

The inflow in the leg was expressed in ml/min; but the peripheral flow was expressed in ml/min per 100 ml tissue. These values were not wholly comparable: in one case, the total blood flow was recorded with no regard to the volume of the leg; in the other case, the blood flow was related to 100 ml tissue. This fact is clear from Fig. 2, where there are great individual variations in the peripheral flow at the same inflow in the leg.

Earlier studies of the reliability of the strain gauge plethysmograph have been made as a comparison between two indirect methods, i.e. between water plethysmography and strain gauge plethysmography (Whitney 1953, Clarke & Hellon 1957, Buerger *et al.* 1959, Paulev 1969, Dahn & Hallböök 1970). The results from the tests have varied. Dahn & Hallböök found a good correlation between water plethysmography and strain gauge plethysmography if proper consideration was given to the hydrostatic pressure of the water plethysmograph and the segmental range of the strain gauge.

To test the reliability of the strain gauge plethysmograph as described in the beginning, it is essential that the peripheral resistance and the blood flow distribution remain unchanged.

The peripheral flow can be studied by the relation between machine flow/machine pressure. Because this relation was unchanged during the perfusion, it was presumed that the peripheral resistance was unchanged. A so far unpublished study examined the relation between skin and muscle blood flow. The technique used is described elsewhere (V). No proofs of any change in flow distribution were found.

Thus the variations in blood flow recorded with strain gauge plethysmograph depend only on variations of the inflow.

The blood flow, recorded with a water plethysmograph, is related to the part of the extremity enclosed in the plethysmograph. With the help of the circumference of the upper and lower openings of the water plethysmograph, the volume of a frustum of a cone is calculated. This is said to be the volume of the extremity. Divergence of the form of the extremity from that of a frustum of a cone means a risk of obtaining wrong blood flow values.

This risk is more obvious with strain gauge plethysmography, because the increase in the volume is the base for flow calculation, and not as with water plethysmograph, a real volume increase in the part of the extremity enclosed in the plethysmograph. The formula for calculating the blood flow with strain gauge plethysmography is made for a circular segment, where the increase in the volume is uniform. The calf, however, has an elliptic or triangular shape. The circle has the greatest area in relation to the circumference in all known geometric figures. The blood flow in the extremity as calculated with strain gauge could therefore be too big. This, moreover, could be accentuated by a non-uniform increase in the volume, which gives a more pronounced elliptic form.

The experiments showed a good correlation between inflow in the leg and the strain gauge plethysmographic recorded flow. The divergence from the circular circumference and the uniform increase in the volume are too small to affect the validity of the strain gauge plethysmograph.

THE RESISTANCE TO FLOW IN THE VEINS

The relation between blood pressure and blood flow has been studied in many different ways. In human studies, external pressure has been used in order to reduce the perfusion pressure (arterio-venous pressure difference). These studies have shown that the blood flow ceased despite a remaining perfusion pressure (Burton-Yamada 1951). This phenomenon was explained by Burton (1951), by applying the law of Laplace to calculate the tension in the wall of various blood vessels, and stated a hypothesis of critical closing pressure. The critical closing pressure has been attributed to the vasomotor tone of the arterioles.

The vasomotor tone of the arterioles is also normally the main resistance to the blood flow and determines the blood flow. The flow resistance from other vessels, such as capillaries and veins, is usually overlooked. In some special situations, however, the venous flow resistance can hypothetically be ascribed importance; for instance, the good effect of a low position for the foot with gangrene.

In order to evaluate the eventual effects on the venous flow resistance, a special model was designed, which makes it possible to reduce the perfusion pressure either by external pressure or by venous stasis. External pressure reduces the square area of the venous tree, and venous stasis increases the square area. A big square area should reduce the flow resistance, whereas a small one should increase the resistance.

Performance: see Fig. 3.

Comments on the performance:

There are several difficulties in making a reliable model for in vivo studies of the relation between blood pressure and blood flow. One problem, for instance, is the way the blood pressure varies, as to some extent, it can disturb the relation to the blood flow that is to be studied. There are some well-known possibilities to reduce the perfusion pressure.

1. Variation of the central pressure by haemorrhage. These studies can usually only be performed with animals. Even if this type of experiments could be carried out on patients with haemorrhagic shock, they could create an unfavourable disturbance. Lowering the central pressure produces, among other things, a peripheral constriction which reduces the value of recording the flow in the extremity.
2. Variation of the peripheral pressure by clamping artery and veins. Valuable studies can in this way be done only on animals, with a risk of injuring the innervation of the vessels.

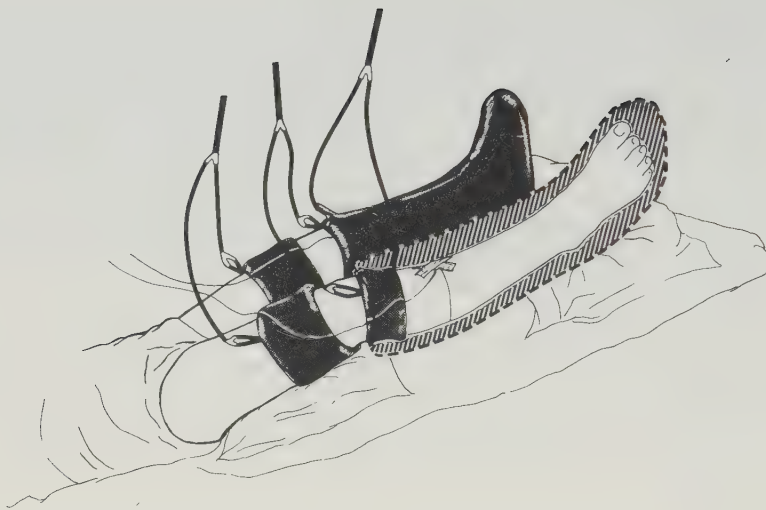


Fig. 3: Flow recording was done with a strain gauge plethysmograph (Whitney 1953) modified according to Hallböök (1970) placed around the calf.

The collection pressure was the same as the diastolic pressure in the arm. The collection cuff was placed just below the knee.

The venous stasis was produced with the aid of a collection cuff above the knee. The pressure of the veins was considered to be the same as that of the cuff (Brown et al. 1966).

External pressure was established by a specially made air splint which enclosed the foot and the lower leg.

Circulatory arrest was produced by the collection cuff below the knee. The circulatory arrest always lasted for 3 minutes.

3. Variation of the peripheral pressure with external pressure. External pressure reduces the transmural pressure, the tensions in the wall of the vessels diminish, which gives bigger volume pulsations, as the pressure pulsations are the same. The effect of volume pulsation on the tone of arterioles is not known. On the venous side, small external pressure reduces the volume of blood. Greater external pressure first causes the veins and then the capillaries to become empty; thereafter the arterial pressure will establish a new equilibrium with a

reduced perfusion pressure and diminished square area of the veins.

4. Variation of the peripheral pressure with venous stasis. Venous stasis dilates the capillaries and veins. The compliance changes, the speed of the blood flow reduces, and filtration of the capillaries increases.

It can be concluded that no really good method exists for studying the relation between blood pressure and blood flow.

We began our experiments with the same type of pressure plethysmograph as Burton-Yamada (1951) described. Pressure plethysmograph is a plethysmograph with which external pressure can be produced, and at the same time, the blood flow can be recorded. We very soon found it necessary to use a tube with a large diameter between the plethysmograph and the volume recorder, because among other things, the volume pulsations otherwise damped down too much. In spite of this arrangement, the pressure plethysmograph was unreliable. We could record a reactive hyperaemia in the pressure plethysmograph when the applied pressure was greater than the systolic blood pressure. Ludbrook & Collins (1967) showed that the transmission of pressure just inside the opening of the plethysmograph is not complete. The same plethysmograph cannot be used for both applying external pressure and recording the blood flow.

The part of the extremity to which the external pressure is applied is smaller than the part from which the flow is recorded. It is impossible to construct a segmental plethysmograph in such a way that this border zone is negligible (Fig. 4).

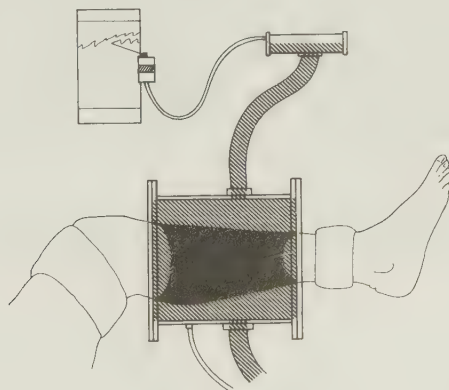


Fig. 4: Pressure plethysmograph. The part of the extremity to which the external pressure is applied is smaller than the part from which the flow is recorded.

We left the pressure plethysmograph and began to use separate arrangements for applying external pressure and recording blood flow. External pressure was first produced by a conventional air splint (Curri 1944, Gardner 1962. It was easy to handle, as in the front, it had a zip-fastener. The transmission of the pressure under the zip-fastener, however, was insufficient. The strain gauge here was badly deformed, and the size of this deformation was dependent on the external applied pressure. We had to design a special air splint without a zip-fastener.

It should be noted that studies have been made with separate transmission of the pressure and flow recording (Roddie-Shepherd 1957, Ashton 1963, Ludbrook-Collins 1967); these confirmed the result of Burton and Yamada.

In the first series of our experiments, a direct comparison was made between the effect on the resting blood flow of the external pressure and the venous stasis. In every case, external pressure reduced the resting blood flow more than the corresponding venous stasis (Fig. 5, 6, 7).

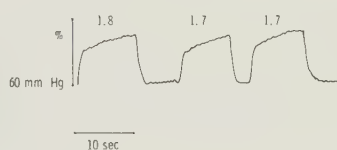


Fig. 5: Resting flow (ml/min x 100 ml tissue) at different external pressures (0, 30 and 60 mm Hg) in one subject.

Fig. 6: Resting flow (ml/min x 100 ml tissue) at varying degree of venous stasis (0, 30 and 60 mm Hg) in one subject.

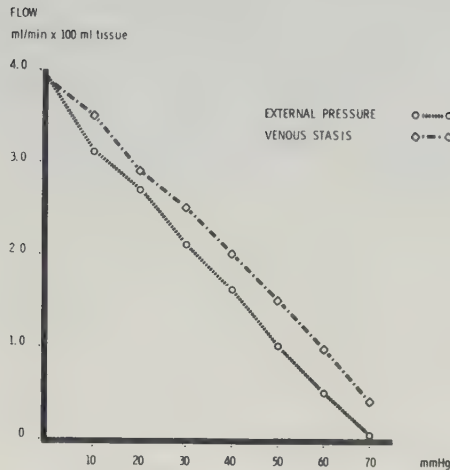


Fig. 7: Comparison between the effect of external pressure and venous stasis on the resting blood flow (ml/min x 100 ml tissue). Mean value for 10 legs from 5 subjects.

In order to exclude a constant error of method and to get more support for the found result, the first series of experiments was completed with another series of experiments. First, an external pressure was applied and the actual resting flow recorded. With the external pressure still applied, a venous stasis, 15 mm Hg greater than the external pressure, was added, and the new resting flow was recorded. Despite the perfusion pressure being reduced by 15 mm Hg, a greater resting flow was found with the combination of external pressure and venous stasis than with external pressure alone (Fig. 8). In the last series of experiments, the difference between the two compared experiments was a venous stasis of only 15 mm Hg, which is why a constant error of method seems less probable as an explanation for the obtained difference.

The difference found between the effect of external pressure and venous stasis of the resting flow can be explained in the following ways:

1. Venous stasis with dilatation of the venous tree gives rise to a dilatation of the arterioles with diminished resistance to flow.
2. External pressure and venous stasis give rise to reduction and dilatation, respectively, of the veins, as described earlier.

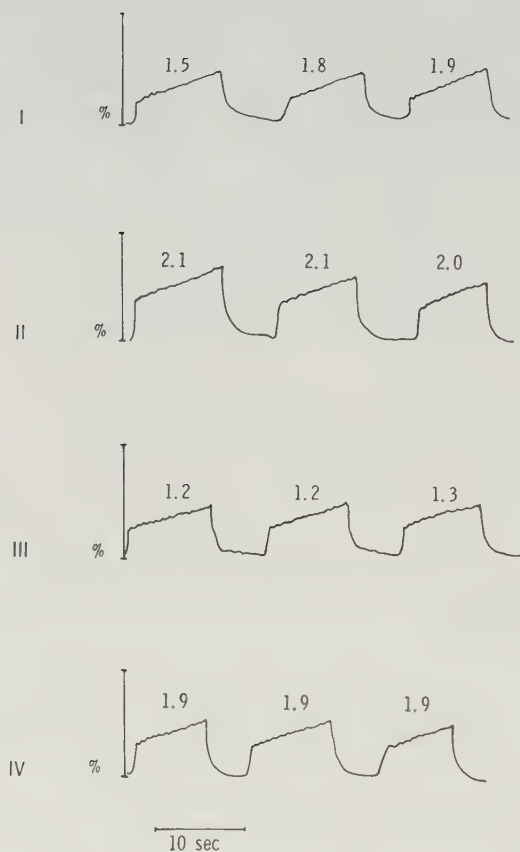


Fig. 8: Effect of external pressure, and external pressure, combined with venous stasis on the resting flow (ml/min $\times$ 100 ml tissue) in one subject.

- I External pressure 30 mm Hg.
- II External pressure 30 mm Hg + venous stasis 45 mm Hg.
- III External pressure 40 mm Hg.
- IV External pressure 40 mm Hg + venous stasis 55 mm Hg.

During reactive hyperaemia, the tone of the arterioles is eliminated; therefore a series of experiments were made to evaluate the above given explanations.

Comparison of the effect of external pressure with that of the venous stasis on the reactive hyperaemia showed that external press-

ure reduced the blood flow more than the corresponding venous stasis did (Table 1).

		EXTERNAL PRESSURE				VENOUS STASIS			
		0	15	30	45	0	15	30	45
MEAN VALUE	FF	19	11	8	5	19	15	12	10
	PF	22	15	11	6	22	16	13	10
S.D.	FF	5,6	2,6	1,9	1,3	5,6	4,6	3,8	3,3
	PF	4,9	3,0	2,3	1,3	4,9	4,0	3,1	3,6

Table 1: Mean values of first flow and peak flow at different external pressure and venous stasis (0, 15, 30, 45 mm Hg) from 10 legs (5 subjects).

A series of experiments was also made, first with external pressure alone, and then in combination with venous stasis 15 mm Hg greater than the external pressure during reactive hyperaemia. The combination of external pressure and venous stasis gave a greater blood flow than external pressure alone, despite a reduction of the perfusion pressure (Fig. 9).

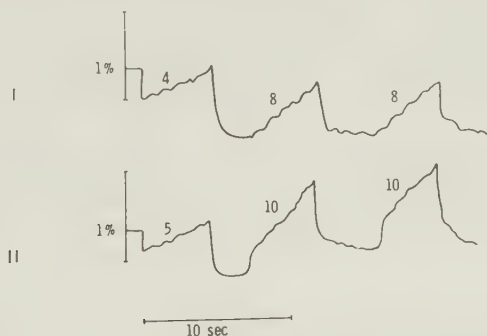


Fig. 9: Reactive hyperemi from one leg. Blood pressure: 120/75. Collection pressure: 75.

- I External pressure 30 mm Hg.
- II External pressure 30 mm Hg and 45 mm Hg venous stasis.

The difference found between the effect of external pressure and that of venous stasis on the resting blood flow was also found during reactive hyperaemia. This means that the resistance to flow in the veins under special circumstances is of importance.

A comparison similar to that made by us has been reported by Roddie & Shepherd (1957). They examined the resting blood flow through the finger during external pressure, venous stasis, and elevation of the finger. The blood flow was estimated from heat elimination with thermocouples. Even small external pressure and moderate elevation reduced the resting blood flow in the finger. Venous stasis, on the other hand, had no effect on the blood flow, until it reached 30-60 mm Hg or higher. The experiments gave proofs of critical closing pressure during external pressure and elevation, but not during venous stasis. The difference between the effect of external pressure and that of venous stasis on the resting blood flow was thought to depend on the dilatation of the veins during venous stasis, which gave rise to a dilatation of the arterioles and so reduced the resistance to flow.

Our experiments gave similar results, which means that external pressure reduced the blood flow more than venous stasis did. Our interpretation of the results differed, however, from that given above. Our reasons for this are the results found during reactive hyperaemia.

During these experiments, we found a corresponding difference between external pressure and venous stasis, both during reactive hyperaemia and during resting blood flow, which means that the resistance to the flow in the veins is of importance.

These experiments cannot determine whether it is the external pressure that mainly increases the resistance to flow or the venous stasis that reduces the resistance to flow, or a combination of both. To reach a decision, it is necessary to reduce the perfusion pressure by external pressure, by venous stasis, and by some other method that has no influence on the venous resistance.

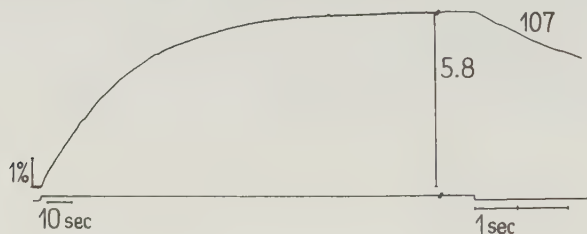
We will point out, however, that during experiments with external pressure, concerning blood pressure and blood flow, regard must be paid to the resistance to venous flow. Critical closing pressure can perhaps to some extent be explained as a result of the resistance to the venous flow.

The performed experiments cannot determine whether the changes in the resistance to the venous flow during external pressure and venous stasis have any clinical application. Some circulatory problems, however, could be explained based on our data. Patients with gangrene in a foot get a better flow and reduction of pain when the foot is lowered outside the bed. In a patient with gangrene, arterioles are maximally dilatated. Lowering the foot cannot in-

crease the perfusion pressure, but it increases the pressure in arteries as well as in veins. The increased venous pressure dilates the venous tree and gives an increased square area. The increased square area diminished the flow resistance.

STUDIES OF DEEP VENOUS THROMBOSIS

At the hospital in Lund (Dahn-Eiriksson 1968; Hallböök-Göthlin 1971; Hallböök-Ling 1973) and also elsewhere (Sachaguchi *et al* 1970; Bygdeman *et al* 1972) plethysmographic diagnosis of deep venous thrombosis has been shown to be a simple and reliable method. This plethysmographic diagnosis is based on venous volume (i.e. the increase in the volume of the extremity, which occurs when a collection pressure is applied until equilibrium is established), and venous emptying (i.e. the fastest decrease in the volume of the extremity, which occurs when a collection pressure is suddenly released after measurement of the venous volume).



NORMAL LEG



THROMBOTIC LEG

Fig 10: Plethysmogram from a patient with a normal and a thrombotic leg.

Measurement of venous volume and venous emptying is made with a strain gauge plethysmograph (Whitney 1953) modified according to Hallböök *et al* (1970). The strain gauges are placed around calf and ankle. The collection pressure is 50 mm Hg. The collection cuffs are placed above the knee. The diagnosis of deep venous thrombosis was made according to the following criteria (Hallböök-Göthlin 1971):

Calf: Venous emptying: less than 35 ml/min x 100 ml tissue.

Venous volume: less than 1.9 ml/100 ml tissue not decisive, but lends considerable support to the diagnosis.

Ankle: Venous emptying: less than 30 ml/min x 100 ml tissue.
Venous volume: not conclusive:

In the following, two problems are discussed; namely, the cause of the reduced venous volume and slower venous emptying and the resting blood flow of the deep venous thrombosis.

The reason for a reduced venous volume and a slower venous emptying at deep venous thrombosis has been discussed (Dahn-Eiriks-son 1968), but has not been further studied. By comparing the effect of high position with low position and comparing the effect of various external pressures at low position, the above mentioned changes have been studied.

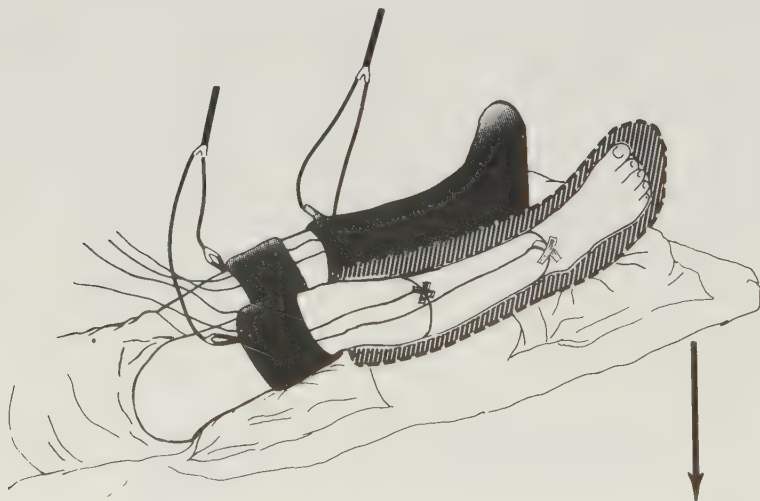


Fig 11: Experimental set up. Cuffs above the knees for producing collecting pressure. Double-walled bootshaped inflatable bags on the feet and lower legs for applying external pressure. Strain gauges on calves and ankles for flow determination.

The effect of high position compared with low position and the effect of external pressure in low position are about the same. Both high position and low position increase the emptying of the veins before the measurement starts, reduce the effective collection pressure, and increase the pressure gradient distal-proximal of the strain gauge. A relatively good agreement is thus to be expected between the effect of external pressure and elevation. As the effect of external pressure on the venous resistance is the same bilaterally, no correction for this has been made.

The experiments have been made in 12 patients with one thrombotic and one normal leg.

The venous volume of the calf was greater in the normal than in

the thrombotic leg, and the difference increased with high position and external pressure of 10 mm Hg. According to the venous volume in the ankle, no difference was found between the normal and the thrombotic leg. During high position and external pressure, it was the same.

VENOUS VOLUME OF THE CALF

				<u>NORMAL</u>			<u>THROMBOTIC</u>		
				$\bar{x}$	S.D	n	$\bar{x}$	S.D	n
L.P	EP	0		1,9	0,48	12	1,2	0,34	12
L.P	EP	10		2,7	0,84	12	1,0	0,30	12
L.P	EP	20		2,4	0,96	12	1,0	0,38	12
L.P	EP	30		1,4	0,69	12	0,8	0,41	12
H.P	EP	0		3,0	0,92	12	1,4	0,38	12

Table II: Mean value of the venous of volume of the calf (100 ml/min). LP means low position and HP high position. EP means external pressure produced by the double-walled bootshaped inflatable bag.

VENOUS VOLUME OF THE ANKLE

				<u>NORMAL</u>			<u>THROMBOTIC</u>		
				$\bar{x}$	S.D	n	$\bar{x}$	S.D	n
L.P	EP	0		1,4	0,40	12	1,2	0,33	12
L.P	EP	10		1,7	0,73	12	1,2	0,40	12
L.P	EP	20		1,4	0,61	12	1,1	0,37	12
L.P	EP	30		0,8	0,47	12	0,7	0,41	12
H.P	EP	0		2,1	0,67	12	1,8	0,50	12

Table III: Mean value of the venous volume of the ankle (ml/100 ml tissue) abbreviations see table II.

Venous volume is directly dependent on the venous pressure (Dahn-Eiriksson 1968). The relation is not linear. The volume increase becomes less pronounced when the collection pressure is more than 50 mm Hg.

The low venous volume measured at deep venous thrombosis could be the result of:

1. increased venous pressure
2. increased tissue pressure
3. space-occupying thrombotic masses

Ad 1. The collection pressure will be reduced by an increased venous pressure. If there was an increased venous pressure, it must be distal to the thrombosis. A difference between normal and thrombotic ankle should then be found, but no such difference was found.

An increased venous pressure should give a decreased resting blood flow according to our experiments (II + III), but the resting flow is unchanged or increased (V).

It thus does not seem probable that an increased venous pressure plays any role in the reduced venous volume (Christensen 1967).

Ad 2. The effect of the collection pressure is reduced if the tissue pressure increases, i.e. the oedema of the thrombotic leg. External pressure gives an increased tissue pressure, but influences the venous volume by emptying the veins before the measurement. From unpublished experiments with regional perfusion, however, we know that the oedema must be at least 10-15 % of the circumference to have any effect on the venous volume.

Ad 3. The result demonstrates that the venous volume in the thrombotic leg does not increase during elevation or external pressure, whereas that in normal legs does. Elevation and external pressure result in a more complete emptying of the veins before the start of measurement during normal conditions. The thrombotic masses must be why that elevation and external pressure have no effect on the venous volume in the thrombotic leg, because the effective collection pressure was about the same.

The thrombotic masses have a space-occupying effect and are fixed at the walls; external pressure and elevation have no effect on them.

In normal legs, the venous emptying will be faster if external pressure or elevation is added, but no such effect is seen in the thrombotic leg.

VENOUS EMPTYING OF THE CALF

				<u>NORMAL</u>			<u>THROMBOTIC</u>		
				$\bar{x}$	S.D	n	$\bar{x}$	S.D	n
L. P	EP	0		49	11,2	12	20	6,6	12
L. P	EP	10		65	20,6	12	17	6,9	12
L. P	EP	20		78	30,2	12	16	5,8	12
L. P	EP	30		79	32,9	12	14	5,7	12
H. P	EP	0		60	12,2	12	20	5,6	12

Table IV: Mean value of the venous emptying of the calf (ml/100 ml tissue x min).

VENOUS EMPTYING OF THE ANKLE

				<u>NORMAL</u>			<u>THROMBOTIC</u>		
				$\bar{x}$	S.D	n	$\bar{x}$	S.D	n
L. P	EP	0		32	9,8	12	23	6,3	12
L. P	EP	10		35	10,4	12	17	5,4	12
L. P	EP	20		36	11,0	12	15	4,7	12
L. P	EP	30		25	12,1	12	12	4,1	12
H. P	EP	0		41	9,4	12	22	5,5	12

Table V: Mean value of the venous emptying of the ankle (ml/100 ml tissue x min).

External pressure and elevation increase the venous volume in the normal leg, but have no effect on the thrombotic leg. It seems reasonable to suppose that venous emptying is related to venous volume.

Patients with deep venous thrombosis have a reduced venous volume and a slower venous emptying, whereas patients with vari-

cose veins have a larger venous volume and a faster venous emptying (Dahn-Erikson 1968). Eiriksson-Dahn (1968a) in a material of 9 thrombotic legs, however, found no correlation between venous volume and venous emptying. Bygdeman *et al* 1972 found a good correlation between venous volume and venous emptying in normal and in varicose legs. The results found here confirm the above findings.

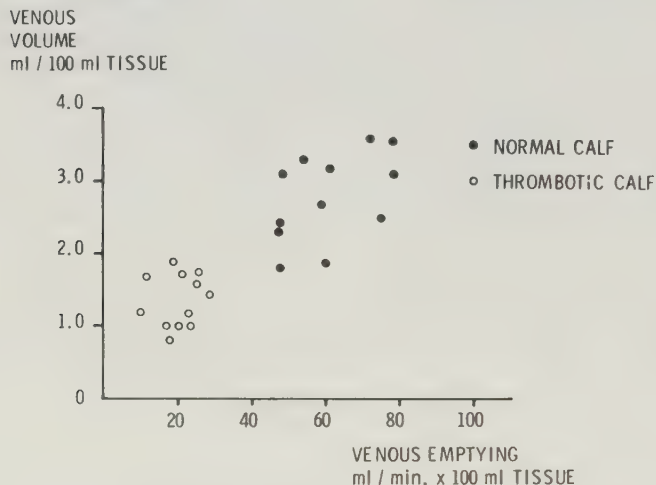


Fig 12: Relation between venous volume (ml/100 ml tissue) and venous emptying (ml/100 ml tissue x min) in high position in the calves of patients with a deep venous thrombosis in one leg and the contralateral normal.

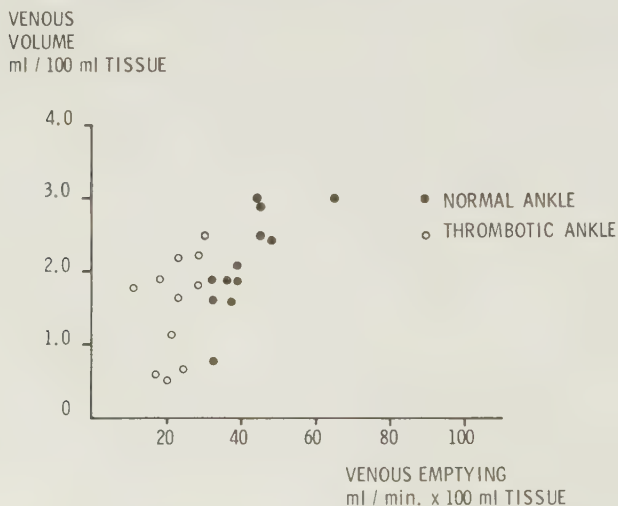


Fig 13: Relation between venous volume (ml/100 ml tissue) and

venous emptying ($\text{ml}/100 \text{ ml tissue} \times \text{min}$) in high position in the ankle of patients with a deep venous thrombosis in one leg and the contralateral normal.

I.e. that a good correlation exists between the venous volume and the venous emptying in a normal leg, but not in a thrombotic leg.

External pressure and elevation increase the pressure gradient distal-proximal. As this pressure gradient is the same in the normal leg as in the thrombotic leg, it cannot explain the difference.

It seems probable that the slower venous emptying of the thrombotic leg is related to a reduced outflow. The reduced outflow will be an effect of the space-occupying masses fixed at the walls of the vessels.

The reduced venous volume and slower venous emptying seems thus to result from the space-occupying effect of the thrombotic masses.

This means that thrombosis situated distal to the strain gauge might have both normal venous volume and venous emptying; thrombosis situated proximal to the strain gauge might have normal venous volume.

II.

The resting blood flow at deep venous thrombosis of the leg has been found to be moderately increased or normal, as recorded with water plethysmography (Dahn-Eiriksson 1968). In order to study more closely the resting blood flow at deep venous thrombosis, resting blood flow has been determined with strain gauge plethysmography at calf and ankle. The resting blood flow, however, varies greatly even in normal circumstances between different individuals; therefore the measurements have been made in patients with thrombosis in one leg and with the other normal, the latter being used as a control.

The material consisted of 34 patients, of whom 3 had a distal thrombosis (Hallböök-Göthlin 1971). Distal thrombosis has a pathological venous emptying of the ankle, but otherwise normal values. These 3 patients are discussed separately. We found that resting blood flow in the calf was the same in the normal and the thrombotic leg, but in the ankle, it was greater in the thrombotic than in the normal leg.

	Calf			Ankle		
	$\bar{x}$	S.D.	N	$\bar{x}$	S.D.	N
<i>Resting blood flow</i>						
Normal leg	2.8	1.03	31	1.2	0.60	31
Thrombotic leg	3.1	1.19	31	2.8	1.24	31
<i>Venous emptying</i>						
Normal leg	67	14.9	31	45	13.3	31
Thrombotic leg	24	8.71	31	22	12.6	31
<i>Venous volyme</i>						
Normal leg	3.0	1.47	31	2.2	0.80	31
Thrombotic leg	1.6	0.76	31	1.7	0.78	31

Table VI: Mean value for resting flow (ml/min $\times$ 100 ml), venous emptying (ml/min $\times$ 100 ml) and venous volume (ml/100 ml) in 31 patients with acute thrombosis in one leg and the other normal.

The quotient ankle/calf resting blood flow was always greater in the thrombotic leg than in the normal leg.

The 3 patients with distal thrombosis had no increased resting blood flow in the ankle compared with the normal leg.

Name, sex, age	Normal calf	Normal ankle	Thrombotic calf	Thrombotic ankle	Quotient normal ankle/calf	Quotient thrombotic ankle/calf
<i>Distal thrombosis</i>						
VO, man, 42 years	4.5	1.4	5.9	1.5	0.3	0.3
LL, woman, 19 years	2.5	0.9	2.1	0.3	0.4	0.2
ÅA, man, 54 years	7.0	0.8	6.0	0.5	1.1	0.8

Table VII: Resting flow (ml/min x 100 ml) in 3 patients with an acute thrombosis in the distal part of one leg and the other leg normal.

The blood flow through the calf and ankle consists of skin and muscle blood flow. By utilizing the fact that different parts of the leg consist of different proportions of skin and muscle, an attempt was made to study the skin blood flow. The blood flow in the ankle segment consists of skin blood flow to a greater extent than that in the calf segment, where the muscle component is dominant. If the skin blood flow of the leg increases, it follows that the flow in the ankle segment should increase more than that in the calf. Our results show a significant increase in the resting blood flow in the ankle of the thrombotic leg, which means an increased skin blood flow, according to the above discussion.

The most probable cause of the increased skin blood flow seems to be that the deep venous thrombosis obstructs the outflow through the deep veins. The blood therefore goes through the superficial veins which become dilated (Huntersign). The increased blood flow in the superficial veins causes an increase in temperature (Provan's sign).

The distal thrombosis does not obstruct the outflow enough to cause an increase in the flow in the superficial veins.

It has been shown earlier (II + III) that venous stasis reduces the resting flow. At deep venous thrombosis, however, we found a normal resting blood flow, which is why the venous pressure does not seem to be increased. The serious changes in circulation which sometimes appear at deep venous thrombosis (cerulens) could

thus be explained by an increased venous pressure which reduces the resting flow.

CONCLUSIONS

A linear relation has been found between inflow of the blood in the leg and the peripheral blood flow measured with strain gauge. Despite divergences from the circular form of the calf and the non-uniform increase in the calf when collection pressure is applied, reliable recordings can be made with the plethysmograph.

Reduction of the perfusion pressure by external pressure or venous stasis reduces the resting blood flow as well as that during reactive hyperaemia. The reduction during external pressure is greater than during venous stasis. The probable cause of this difference seems to be that the resistance to flow in the veins during external pressure differs from that during venous stasis. With changes in the resistance to flow in the veins, the phenomena can be explained as the good effect of low position of the gangrenous extremity.

The plethysmographic manifestations of the deep venous thrombosis, i.e. increased skin blood flow, the reduced venous volume, and the slower venous emptying, all seem to be a manifestation of the space-occupying effect of the thrombotic masses.

The resting flow at deep venous thrombosis is normal or increased. An increased venous pressure (cf. venous stasis) gives a reduction of the resting flow (II, III). It seems possible that the venous pressure is not increased at deep venous thrombosis. The serious changes of circulation sometimes seen at deep venous thrombosis (cerulens) could be explained by a greater venous pressure causing a reduced resting flow.

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